

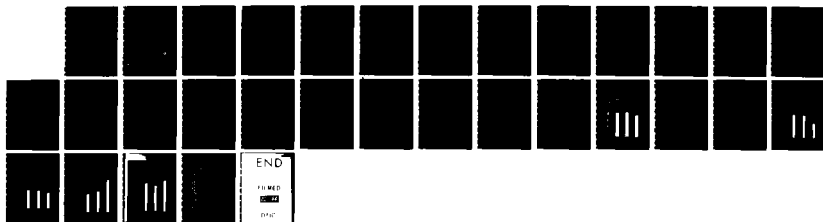
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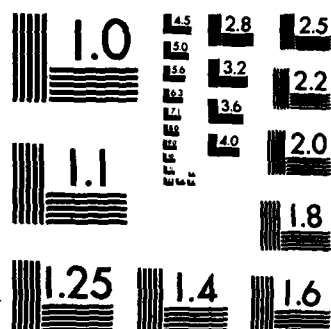
EXERCISE IN THE HEAT EFFECTS OF SALINE OR BICARBONATE
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plasma osmolality. Lactate levels were significantly increased in all three groups with no notable inter-group differences. While venous (v) blood pH and bicarbonate levels were decreased following exercise in the SAL and CON groups, they were unchanged in the BIC group. While $vPCO_2$ was unaffected by fluid administration in all three groups, vPO_2 was significantly increased following exercise in the heat in all groups. We concluded from these experiments that while BIC infusion prevented the acidosis and hypobicarbonatemia induced by exercise in the heat to hyperthermic exhaustion, no beneficial effects on physical performance or thermoregulation ensued.

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**Exercise in the Heat:
Effects of Saline or Bicarbonate Infusion**

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ABSTRACT

Adult, male rats (n=17/group, 300-320 g, physically untrained) were exercised (9.14 m/min) in the heat (35°C) to hyperthermic exhaustion ($T_{re} = 43^{\circ}\text{C}$) after infusion of 2 ml of 7.5% sodium bicarbonate (BIC) or 2 ml of 0.9% sodium chloride (SAL). BIC or SAL administration had no effects on endurance when compared with rats receiving no exogenous fluid (CON) while the rate of heat gain was significantly increased in the BIC-treated group. Following exercise, the BIC group manifested significantly decreased hematocrit and plasma protein levels, but exaggerated increments in plasma osmolality. Lactate levels were significantly increased in all three groups with no notable inter-group differences. While venous (v) blood pH and bicarbonate levels were decreased following exercise in the SAL and CON groups, they were unchanged in the BIC group. While $v\text{PCO}_2$ was unaffected by fluid administration in all three groups, $v\text{PO}_2$ was significantly increased following exercise in the heat in all groups. We concluded from these experiments that while BIC infusion prevented the acidosis and hypobicarbonatemia induced by exercise in the heat to hyperthermic exhaustion, no beneficial effects on physical performance or thermoregulation ensued.

Key Words: Hyperthermic exhaustion; hematocrit; acidosis; heat/exercise injury.

INTRODUCTION

During development of heat stroke in dogs by passive exposure to hot environmental conditions, Magazanik et. al.(19) demonstrated an initial respiratory alkalosis followed by a marked metabolic acidosis characterized by lactic acid accumulation and bicarbonate depletion. In an earlier study using heat/exercise injured rats, we (9) had reported that following exercise in the heat to hyperthermic exhaustion, there was observed an inverse correlation between lactate levels and survival time. In fact, one of the most consistent clinical chemical manifestations of heat/exercise injury in our exercising, heat-stressed rat model has been a significant elevation in circulating lactate levels (7,10,11). In their extensive reviews on the mechanisms of heat stroke both Shibolet et. al. (24) and Knochel (15) identified metabolic acidosis as a clinical feature of heat stroke, especially when physical activity was an etiologic factor in development of the injury. Therapeutic intervention in the treatment of heat stroke ordinarily includes administration of fluids or buffers not only to replenish or to expand plasma volume but also to offset the metabolic acidosis which usually accrues (3,13,25).

Many reports have addressed the development and fate of the lactacidemia and ketonemia occurring during exercise even when the work was performed in the absence of environmental stress. For example, Brooks et. al. (2) demonstrated in fasted rats that following exhaustive exercise most of the circulating lactic acid was rapidly oxidized and expired as CO_2 , and not utilized in glycogen synthesis. Asmussen et. al. (1) presented data which indicated that peak lactate levels following bicycle ergometer exercise in humans were correlated with initial

glycogen content at storage sites. The roles of diet, age, and physical conditioning in the development of lactacidemia and ketonemia were investigated by Koeslag et. al. (16) who reported that reduced physical conditioning and increasing age contributed to lactacidemia and ketonemia, respectively. It is generally agreed that highly trained individuals can exercise at a level close to their maximal oxygen consumption without manifesting lactacidemia (6). These workers (6) demonstrated that well-trained distance runners were able to run at nearly 70% of their $\dot{V}O_2$ max before the onset of plasma lactate accumulation; this level of oxygen consumption is considerably higher than was observed in physically unfit subjects (4).

These relationships between either fatigue, lack of fitness, age, or inadequate dietary carbohydrate and the accumulation of circulating lactic acid have prompted several investigators to examine the effects of preinduced alkalosis on physical work performance. Kindermann et. al. (14) demonstrated that infusions of either bicarbonate or Tris-buffer reduced the level of acidemia in human test subjects after exercise, but had no significant effects on performance. Jones et. al. (12) reported that alkalosis induced by oral administration of bicarbonate increased endurance at 95% of maximal power output. Using in vitro techniques, Mainwood and Cechetto (20) demonstrated that, following fatigue, diaphragm strips recovered full tension rapidly and completely in the presence of 25 mM bicarbonate; recovery was delayed and reduced when 2 and 10 mM bicarbonate solutions were utilized. In a recent report Wilkes et. al. (27) concluded that orally administered bicarbonate had beneficial effects on trained middle distance runners.

In our own work (9-11) we have used a combination of light exercise and hot environmental conditions to induce hyperthermic exhaustion and possible heat injury in rats. As a result of the potential benefit of neutralizing acidemia in the treatment of heat stroke and the ergogenic advantages that may result from preinduced alkalosis, we have examined the effects of bicarbonate infusion on the ability to work in the heat. In these experiments we have examined the thermoregulatory, physical, physiological, and hematological sequelae of bicarbonate administration to obtain a comprehensive profile of its effects.

METHODS

Adult, male rats (Sprague-Dawley, Charles River Breeding Laboratories, Wilmington, MA) were housed singly in wire-bottomed cages at an environmental temperature of $21.0 \pm 1^{\circ}\text{C}$. Food (Ralston Purina Rodent Chow) and tap water were available ad lib, and an automatic timing device controlled fluorescent lighting (on, 0600-1800 h). Untrained rats were maintained under these conditions for approximately one week before experimentation to accustom the animals to handling and weighing procedures as well as to entrain the normal diurnal/nocturnal periodicities of body temperature. It was unnecessary to train the rats since they will readily run at the slow treadmill speeds selected for these experiments.

Three groups of animals ($n = 17/\text{group}$) were used for the first experiments. On the day prior to an experiment each rat was fitted with an indwelling Silastic catheter in the external jugular vein for rapid and convenient blood sampling as well as for intravenous administration of the appropriate fluid. The catheter was inserted under sodium pentobarbital anesthesia (50 mg/kg) under aseptic conditions, and had no detrimental effects on the subsequent ability to exercise in the heat. On the following day each animal was fitted with a rectal probe (model #701, Yellow Springs Inst. Co., Yellow Springs, OH) inserted 6 cm beyond the anal sphincter and a surface probe (Yellow Springs #709) secured midlength on the tail to monitor rectal (core) and tail-skin temperatures.

Prior to exercise in the heat a pre-run blood sample (0.7 ml) was obtained to establish normal or control levels of the variables of interest. Hematocrit levels were immediately established and the remaining blood sample was centrifuged (10,000 g, 4°C , 10 min). An aliquot of plasma was removed, and plasma osmolality

was quantitated (Micro Osmometer, Precision Systems, Inc.). The remaining plasma sample was frozen (-20°C) for subsequent analysis of lactate and protein levels. These spectrophotometric procedures were executed on a Gilford Stasar III semi-automated spectrophotometer using test kits prepared by Sigma (lactate) and Worthington (protein).

Following acquisition of control temperatures and the pre-run blood sample, the rats were rapidly removed to a large (3m x 4m x 2m) stainless steel chamber maintained at $35.0 \pm 0.5^{\circ}\text{C}$ (25-30% RH). The rats were run (level treadmill, 9.14 m/min) to hyperthermic exhaustion ($T_{re} = 43^{\circ}\text{C}$, animal unable to right itself). During the treadmill run 2.0 ml of 7.5% sterile sodium bicarbonate solution was infused intravenously into the first group of animals (BIC, $n = 17$, mean wgt = 310.8 g); the infusion required approximately 15 min to complete. The second group of rats (SAL, $n = 17$, mean wgt = 311.5 g) received 2.0 ml of 0.9% sodium chloride over the first 15 min of the treadmill run, and the final group (CON, $n = 17$, mean wgt = 315.8 g) received no infusion. Core and skin temperatures were monitored on a minute-by-minute basis during the treadmill interval. Immediately following removal from the treadmill, a second blood sample (post-run) was taken and treated identically as the first. Following removal of the post-run blood sample, the rats were returned to the holding room.

To assess the acid-base status subsequent to bicarbonate or saline infusion and exercise in the heat to hyperthermic exhaustion, it was necessary to utilize three additional groups ($n = 8/\text{group}$) of rats. These animals underwent identical catheterization, infusion, and exercise protocols save for the removal of only 0.4 ml of blood prior and subsequent to exercise in the heat to hyperthermic

exhaustion. These whole blood samples were used to quantitate venous pH, pO_2 , pCO_2 , and bicarbonate levels in the three groups of rats. These assessments, corrected for body temperature, were made on an ABL 2, Acid-Base Laboratory (Radiometer, Copenhagen). It was necessary to use separate groups of rats in these experiments because of the limitations in the volume of the pre-run blood samples. By using the two groups of rats it was not necessary to withdraw more than 0.7 ml of blood from any single animal prior to exercise in the heat.

Statistical analyses were performed by analysis of variance (18) followed by Tukey's t test corrected for multiple comparisons (17). Where appropriate, the non-paired t test was also used; the null hypothesis was rejected at $p < .05$.

RESULTS

Fig. 1 demonstrates that under the conditions of the present experiments, neither bicarbonate nor saline infusion had significant effects on endurance performance. In fact, mean (\pm SEM) endurance capacities for the three groups of rats were: 36.5 ± 1.6 min, CON, 37.2 ± 1.0 min, SAL, and 33.3 ± 1.8 min, BIC; since the minimal difference of the mean values necessary to achieve significance was 4.2 min, it is possible that with an increased n, the BIC-treated rats may have demonstrated a significant decrement. Fig. 2 illustrates that in the BIC-treated rats there occurred significant effects on thermoregulation following BIC infusion and during exercise in the heat. For example, statistical analysis of the data confirmed that at time 0 there were no significant differences in the mean initial Tre among the three groups of rats. Likewise, through 10 min of exercise in the heat, no differences occurred. However, from 15-20 min there was observed a statistically significant ($p < .05$, minimal significance) increment when the mean Tre of the bicarbonate-infused group was compared with either of the other two groups at any of these time intervals. These differences persisted until the rats running for the shortest time in each group were unable to continue. It is interesting to note in Fig. 3 that, while CON and SAL rats manifested parallel rates of tail-skin temperature increments, again the BIC group demonstrated an elevated rate of Tsk gain.

Fig. 4 illustrates that hematocrit levels were significantly ($p < .01$) reduced in the BIC-treated rats following exercise in the heat; this significant reduction in hematocrit was accompanied by a significant ($p < .01$) reduction in circulating protein levels (Fig. 5). Despite the decrease in protein levels in the

BIC-treated rats, Fig. 6 demonstrates that plasma osmolality increments occurring in all three groups post-exercise ($p < .01$) were exacerbated following BIC administration ($p < .01$). Lactate levels (Fig. 7) were significantly ($p < .01$) elevated following exercise in the heat in all groups post-exercise, but unaffected by SAL or BIC administration.

The hemodynamic studies (Table 1) executed in the supplementary groups of rats ($n = 8/\text{group}$) provided additional information on the responses to fluid infusion as well as to exercise in the heat to hyperthermic exhaustion. In both the SAL-treated and CON rats exercise in the heat induced a slight ($\text{pH} = -.11$, SAL and $\text{pH} = -.10$, CON, respectively), but significant ($p < .05$, both cases), circulatory acidosis which was neutralized by BIC infusion ($\text{pH} = -.03$, $p = \text{NS}$). Analogous results were noted for bicarbonate levels. Exercise in the heat reduced BIC concentrations from 21.3 ± 0.6 mmoles/l to 16.5 ± 0.9 mmoles/l ($p < .05$) in the CON animals and 22.0 ± 0.7 mmoles/l to 15.6 ± 1.1 mmoles/l ($p < .01$) in the SAL-treated rats. However, BIC administration prevented this reduction (22.4 ± 0.6 mmoles/l, pre-run and 23.4 ± 0.9 mmoles/l, post-run, $p = \text{NS}$). Venous pO_2 was significantly ($p < .05$) increased in all three groups following exercise with no apparent effects of infusion (CON, 42.5 mmHg to 57.5 mmHg; SAL, 40.8 mmHg to 60.9 mmHg; BIC, 36.3 mmHg to 53.9 mmHg). Venous pCO_2 levels were unaffected ($p = \text{NS}$) by either infusion or exercise in the heat to hyperthermic exhaustion.

DISCUSSION

Ordinarily, under the conditions used in the present experiments, adult rats will lose 8-10 g of total body weight during exercise in the heat. The overwhelming majority of this weight loss is through salivary secretion (8). In the current experiments the CON rats (no infusion) lost 8.7 g (mean value) of body weight as water while in the infusion groups the mean weight loss was reduced to 6.5 g (SAL) and 5.8 g (BIC) by the replacement fluids. It is important to note, however, that usually this loss in body water is not accompanied by hemoconcentration (Fig. 4) indicating that plasma volume may be conserved at the expense of intracellular and other interstitial fluids under these acute conditions. Although hydration levels would ordinarily be expected to affect thermoregulation or performance in a hot environment (26,28), neither was improved in the current experiment by replacement of approximately 25% of the water loss. This is probably true because of the acute nature of the current protocol, the fact that plasma volume was apparently not decreased by exercise in the heat even when no infusion was involved, and the inability of the animals to use behavioral techniques for thermoregulation because of the exercise contingency.

Infusion of bicarbonate to offset the anticipated acidosis elicited several noteworthy effects on both thermoregulation and several hematologic variables. Initially, it should be noted that the BIC administration caused significant and consistent increments in colonic temperature at a considerable number of sampling times. Significance was attained in comparison with either the non-infused control group or the group receiving saline. In 2 ml of 7.5% sodium bicarbonate solution are approximately 41 mg Na^+ while 2 ml of 0.9% sodium chloride contain about 7 mg

Na⁺. Myers and Brophy (21) reported that the central administration of excess Na⁺ caused hyperthermia when rats were exposed to an ambient temperature of 22-24°C. This work was extended by Nielsen (22) who demonstrated that body temperatures were elevated in rabbits when hyperosmotic NaCl was administered intravenously. In exercising humans Nielson (23) also reported that oral consumption of hyperosmotic solutions of NaCl also effected increments in body temperature. It is interesting to note that in representative data from her study (23) esophageal temperatures began to diverge after 10 min of exercise and by 15 min there appeared to be significant differences, results very similar to our own. Thus, it is reasonable to conclude that the increased hyperthermia noted in the rats receiving bicarbonate may be the result of a central effect induced by the excessive Na⁺ content of the infused bicarbonate solution. Certainly, tail-skin temperatures indicated no deleterious effects on vasodilation caused by the sodium bicarbonate solution, and thus, no decremental effects on peripheral heat loss through the tail-skin (5,29).

The increments in osmolality subsequent to exercise in the heat to hyperthermic exhaustion were anticipated; the excessive elevation in the BIC-treated rats is undoubtedly the result of the hyperosmolality (1786 mOsm/l) of the BIC solution since both hematocrit and total protein were reduced in this group. It is hypothesized that these significant decrements in hematocrit and total protein levels in the post-run samples arose from acute hemodynamic responses to the hyperosmotic BIC infusion. These responses might include water efflux from the red blood cell mass to the hyperosmotic plasma, from endothelial cells to the plasma, and from interstitial fluid into the vascular system.

Our results indicate that despite approximate two-fold increases in circulating plasma lactate levels following exercise in the heat, blood pH was minimally, but significantly, reduced in the SAL and CON groups. Nonetheless, this mild acidosis had no detrimental effects on endurance capacity under the conditions of these experiments. In both these groups (SAL and CON) the concurrent elevations of lactate and decrements in bicarbonate ion may be the result of homeostatic buffering mechanisms designed to remove excess H^+ ions while bicarbonate was not fully replaced due to lowered renal perfusion or possibly direct hyperthermic injury to the kidney (19). Nevertheless, BIC infusion was fully successful both in preventing the acidosis and in maintaining control or normal bicarbonate levels.

The post-exercise increments in vPO_2 are apparently analogous to those noted in heat-stroked dogs by Magazanik et.al. (19). These workers observed that up to a T_{re} of $41.7^\circ C$ vPO_2 decreased, but reversed with further elevations of T_{re} and actually peaked concomitant with maximal T_{re} , and in the current experiments also the post-exercise blood samples were taken at the time of maximal core temperatures. These increments in vPO_2 may be attributable to decreased tissue extraction and utilization in these severely hyperthermic animals.

We concluded from these studies that while BIC infusion prevented the acidosis and the hypobicarbonatemia induced by exercise in the heat to hyperthermic exhaustion, no beneficial effects on physical performance were noted. In fact, the concentration of sodium ions in the 7.5% sodium bicarbonate solution apparently increased the rates of heat gain in the BIC-treated rats. Further, despite increments in intravascular fluid levels, the BIC infusion effected

exaggerated elevations in the osmolality of the post-exercise plasma samples. It is possible that the acute nature of the heat-exercise conditions of the current experiments precluded significant physiological benefit from the infused fluids.

FIGURE LEGEND

Fig. 1. Effects of saline (2 ml, 0.9%) or bicarbonate (2 ml, 7.5%) infusion on the endurance of rats ($n = 17/\text{group}$, 300-320 g) exercised (9.14 m/min, level treadmill) in the heat (35°C) to hyperthermic exhaustion. Mean values + standard errors of the mean are shown for each group. The solutions were infused over the initial 15 min of the exercise interval; control animals received no infusion.

Fig. 2. Effects of saline or bicarbonate infusion on the colonic temperature response during exercise in the heat to hyperthermic exhaustion. Standard errors of the means are not indicated because in many cases these fell within the range of the symbols used. All conditions are as noted under Fig. 1.

Fig. 3. Effects of saline or bicarbonate infusion on the tail-skin temperature response during exercise in the heat to hyperthermic exhaustion. All conditions are as noted under Fig. 1.

Fig. 4. Effects of saline or bicarbonate infusion during exercise in the heat to hyperthermic exhaustion on hematocrit levels in blood samples obtained immediately prior and subsequent to exercise. Blood samples were taken from permanently implanted catheters in the external jugular vein. All remaining conditions are as noted under Fig. 1.

Fig. 5. Effects of saline or bicarbonate infusion during exercise in the heat to hyperthermic exhaustion on plasma protein levels. All conditions are as noted under Figs. 1 and 4.

Fig. 6. Effects of saline or bicarbonate infusion during exercise in the heat to hyperthermic exhaustion on plasma osmolality. All conditions are as noted under Figs. 1 and 4.

Fig. 7. Effects of saline or bicarbonate infusion during exercise in the heat to hyperthermic exhaustion on plasma lactate levels. All conditions are as noted under Figs. 1, 4, and 5.

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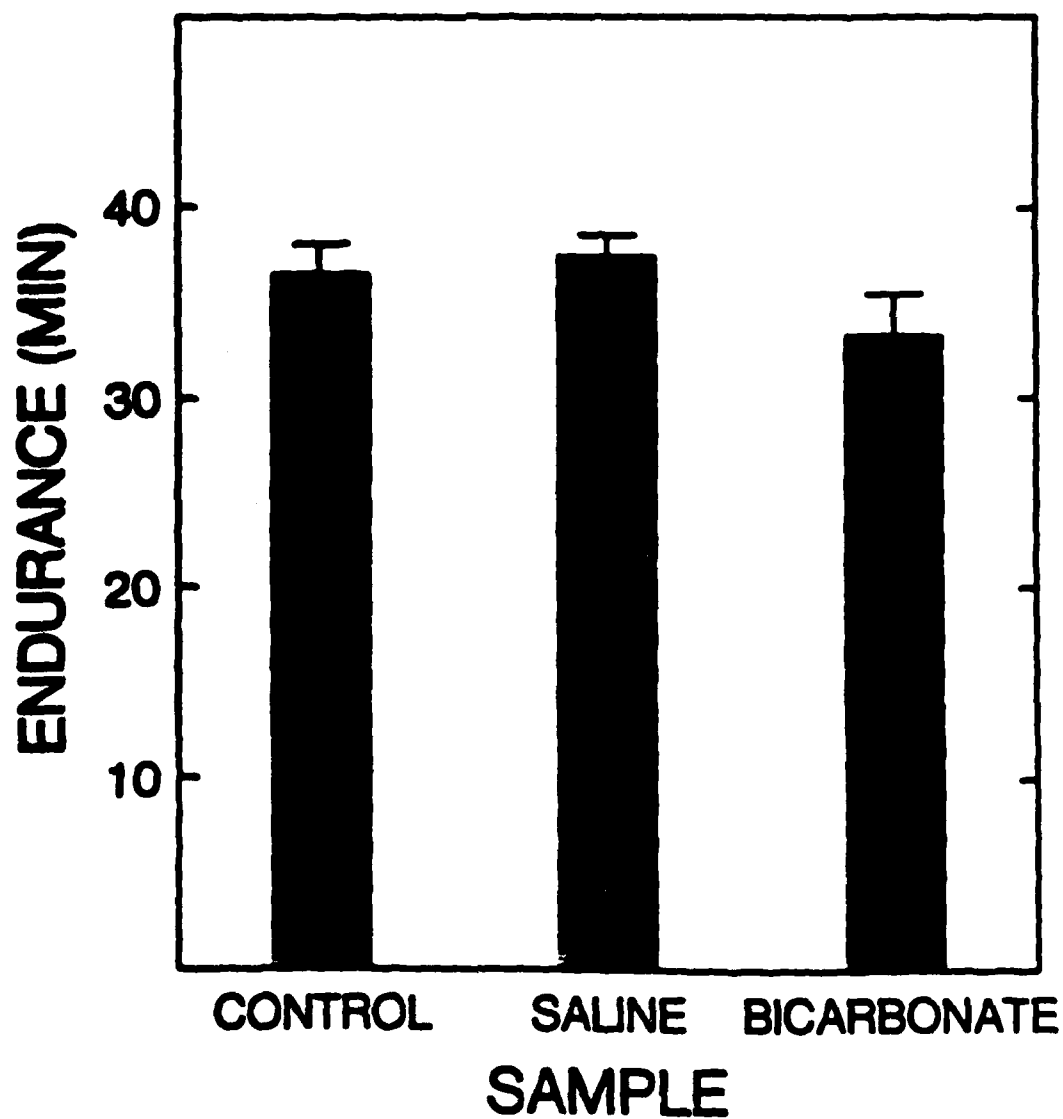
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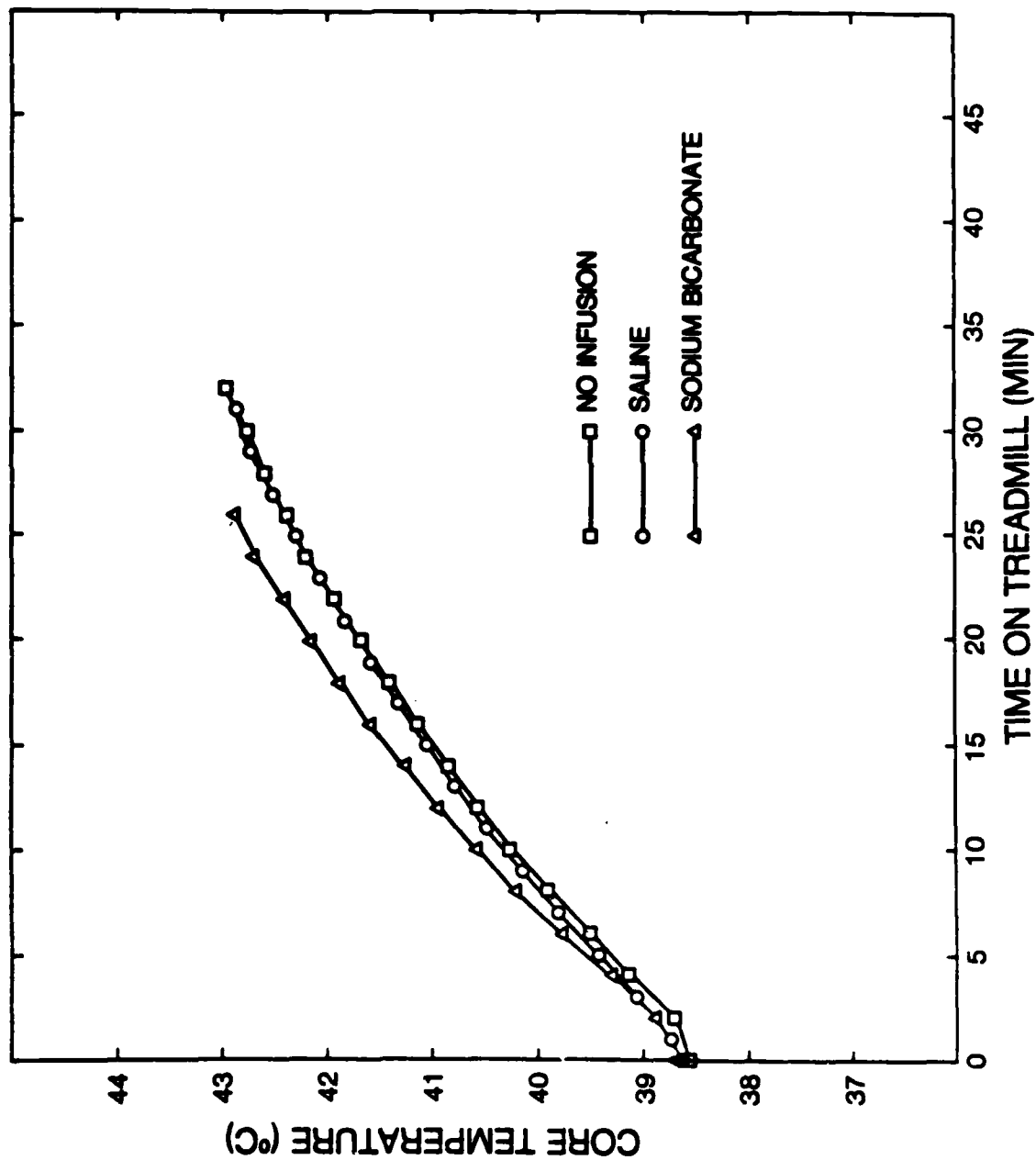
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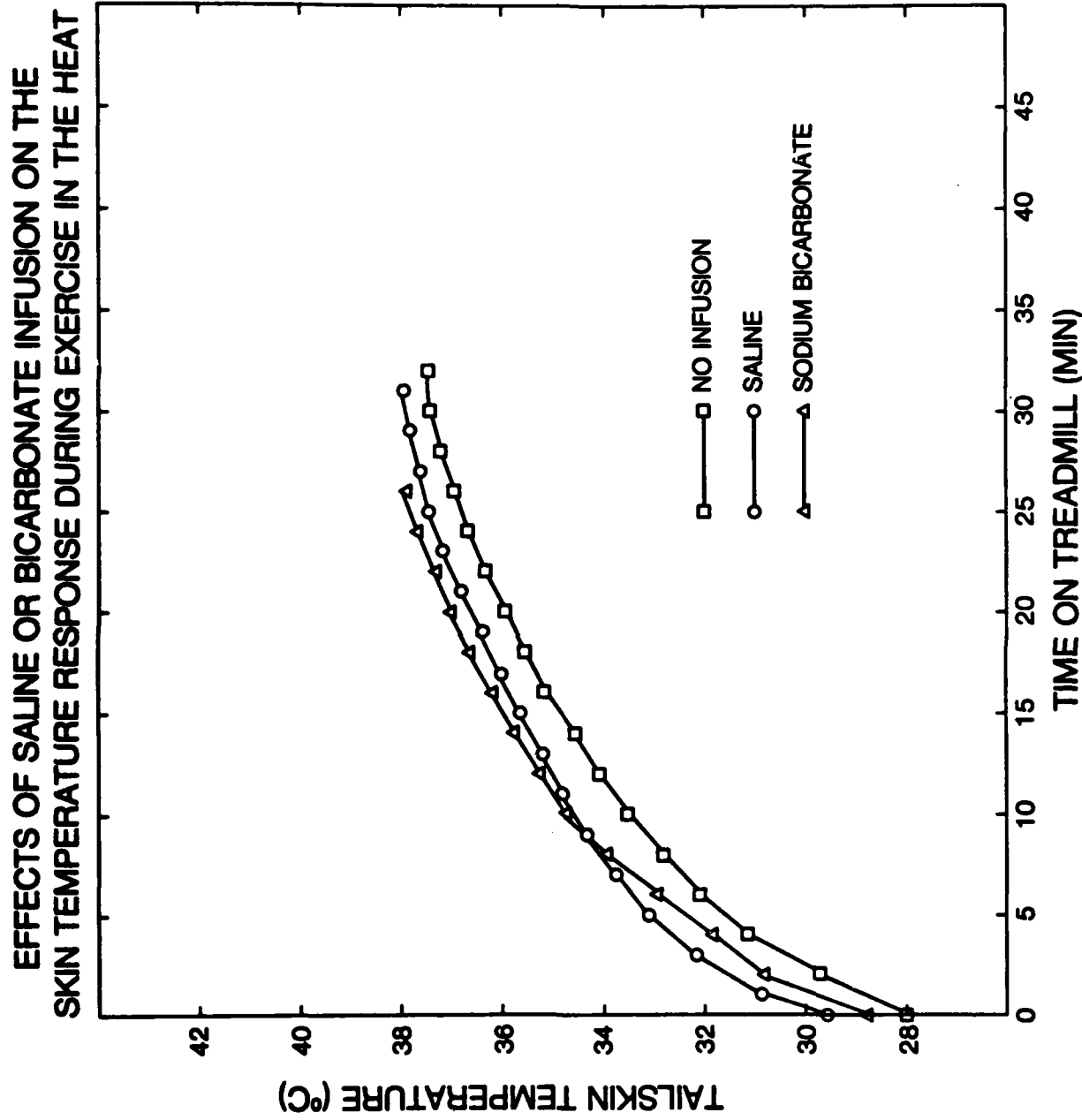
In conducting the research described in this report, the investigators adhered to the "Guide for the Care and Use of Laboratory Animals," as prepared by the Committee on Care and Use of Laboratory Animals of the Institute of Laboratory Animal Resources, National Research Council.

EFFECTS OF SALINE OR BICARBONATE INFUSION ON ENDURANCE OF TREADMILL EXERCISE

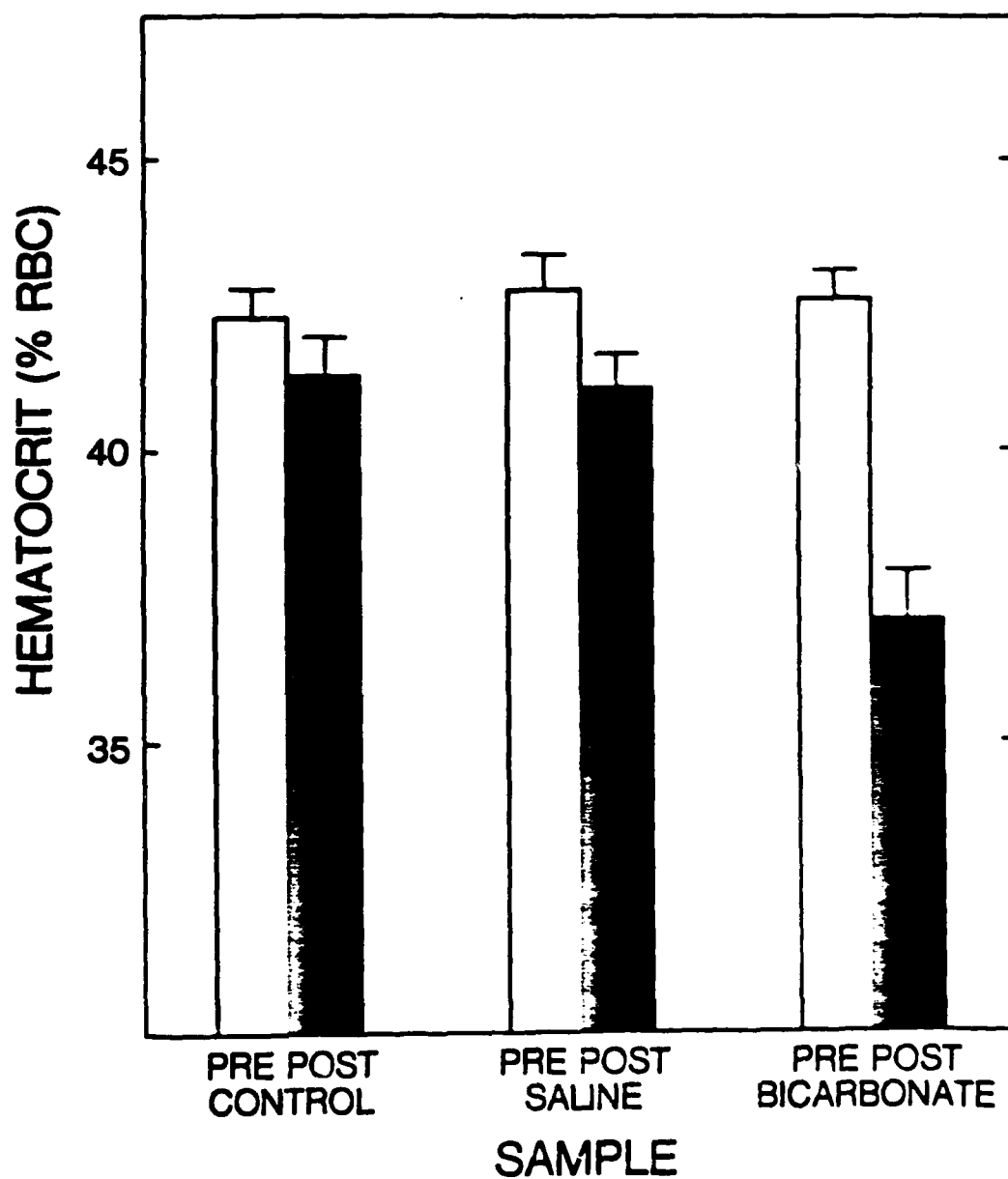


EFFECTS OF SALINE OR BICARBONATE INFUSION ON THE
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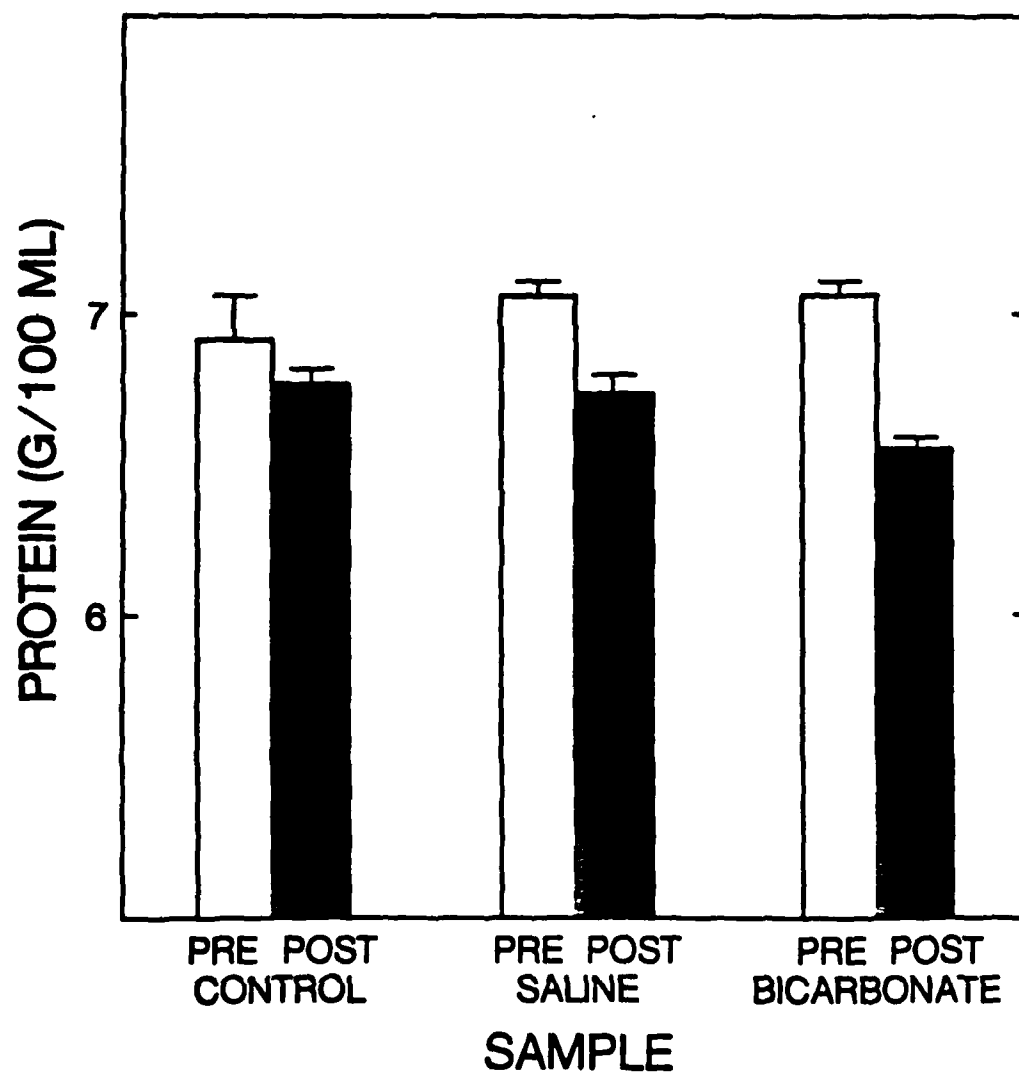




EFFECTS OF SALINE OR BICARBONATE
INFUSION ON HEMATOCRIT LEVELS
BEFORE AND AFTER EXERCISE IN THE HEAT

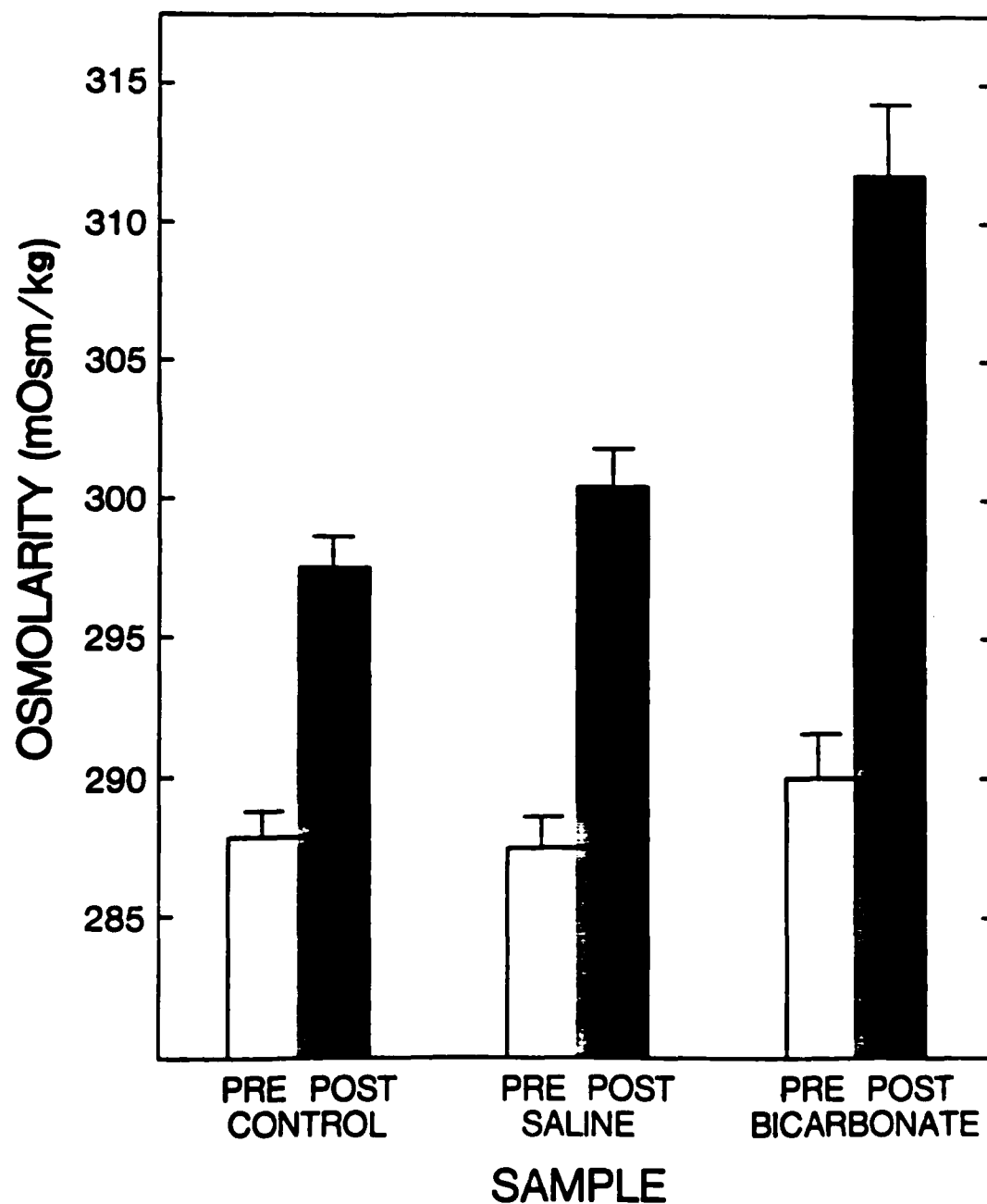


EFFECTS OF SALINE OR BICARBONATE
INFUSION ON PLASMA PROTEIN LEVELS
BEFORE AND AFTER EXERCISE IN THE HEAT

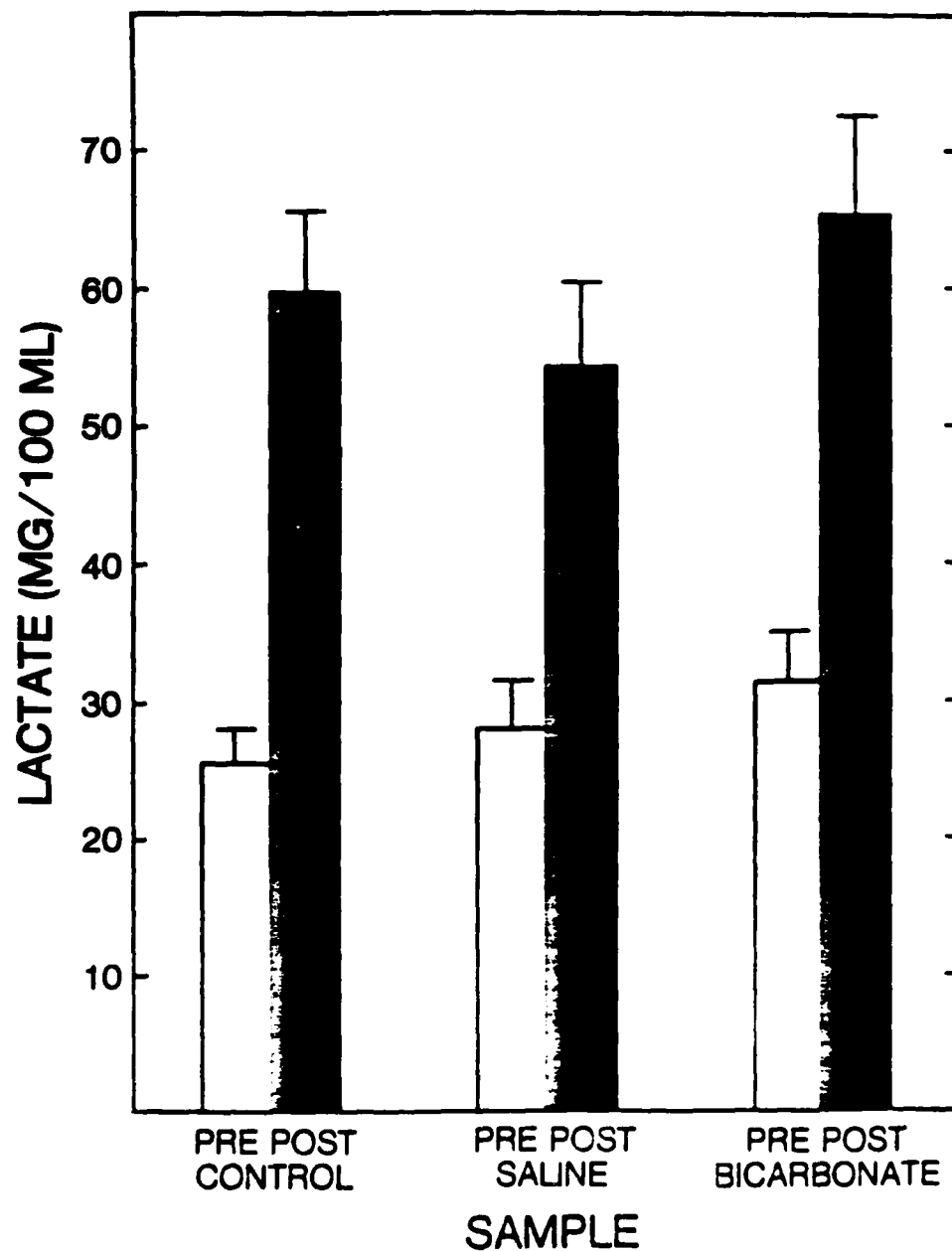


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EFFECTS OF SALINE OR BICARBONATE
INFUSION ON PLASMA OSMOLARITY PREVIOUS
AND SUBSEQUENT TO EXERCISE IN THE HEAT



**EFFECTS OF SALINE OR BICARBONATE INFUSION
ON PLASMA LEVELS OF LACTATE PREVIOUS AND
SUBSEQUENT TO EXERCISE IN THE HEAT**



**TABLE 1. EFFECTS OF SALINE OR BICARBONATE INFUSION
DURING EXERCISE IN THE HEAT ON SEVERAL
HEMODYNAMIC VARIABLES ($\bar{X} \pm \text{S.E.}$)**

| | CONTROL | | SALINE | | BICARBONATE | |
|----------------------------|----------------------|----------------------|----------------------|----------------------|----------------------|---------------------|
| | PRE | POST | PRE | POST | PRE | POST |
| pH | 7.274 ± 0.014 | 7.172 ± 0.031 | 7.300 ± 0.007 | 7.185 ± 0.025 | 7.322 ± 0.009 | 7.294 ± 0.26 |
| HCO ₃ (mM) | 21.31 ± 0.56 | 16.53 ± 0.88 | 22.04 ± 0.70 | 15.59 ± 1.11 | 22.36 ± 0.59 | 23.40 ± 0.94 |
| PO ₂ (mmHg) | 42.50 ± 1.28 | 57.45 ± 0.83 | 40.83 ± 1.48 | 60.94 ± 3.37 | 36.29 ± 2.29 | 53.88 ± 5.41 |
| PCO ₂ (mmHg) | 47.85 ± 1.24 | 50.35 ± 1.72 | 46.54 ± 1.36 | 45.86 ± 2.11 | 45.04 ± 1.07 | 52.16 ± 2.55 |

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02 - 84